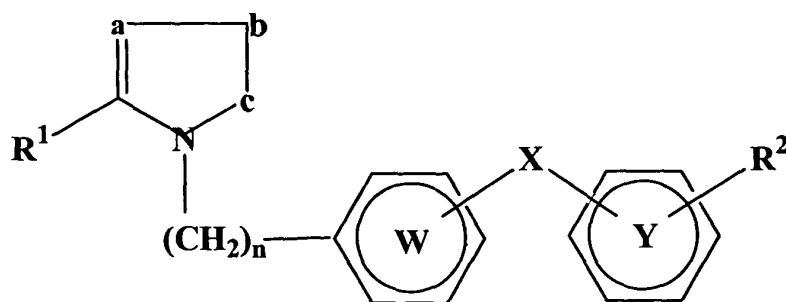


CLAIMS

WHAT IS CLAIMED IS:

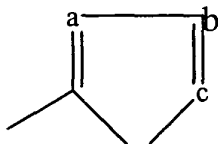
1. A method for treating or prophylactically preventing an inflammatory or metabolic disorder in a mammal by administering to the mammal in need thereof, a therapeutically effective amount of a compound sufficient to (a) at least partially activate peroxisome proliferator activated receptors (PPARs) and (b) at least partially inhibit, antagonize or block an activity of angiotensin II type 1 receptors.
2. The method of claim 1 wherein the treating or prophylactically preventing the inflammatory or metabolic disorder does not cause, promote, or aggravate fluid retention, peripheral edema, pulmonary edema, or congestive heart failure in the mammal.
3. The method of claim 1 wherein the compound is administered in a pharmaceutically acceptable form.
4. The method of claim 1 wherein said compound is administered in a therapeutically effective amount sufficient to prophylactically prevent, slow, delay or treat at least one metabolic disorder or disease selected from the group consisting of insulin resistance, glucose intolerance, impaired glucose tolerance, impaired fasting serum glucose, impaired fasting blood glucose, hyperinsulinemia, pre-diabetes, type 1 diabetes, type 2 diabetes mellitus, insulin-resistant hypertension, the metabolic syndrome, the metabolic hypertensive syndrome, (metabolic) syndrome X, the dysmetabolic syndrome, obesity, visceral obesity, hypertriglyceridemia, elevated serum concentrations of free fatty acids, elevated serum concentrations of C-reactive protein, elevated serum concentrations of lipoprotein(a), elevated serum concentrations of homocysteine, elevated serum concentrations of small, dense low-density lipoprotein (LDL)-cholesterol, elevated serum concentrations of lipoprotein-associated phospholipase (A2), reduced serum concentrations of high density lipoprotein (HDL)-cholesterol, reduced serum concentrations of HDL(2b)-cholesterol, and reduced serum concentrations of adiponectin.
5. The method of claim 1 wherein said compound increases the activity of a PPAR subtype, PPARgamma or a PPARgamma-retinoid X receptor (PPARgamma-RXR) heterodimer.

6. The method of claim 5 wherein the activity of the PPAR subtype, PPARgamma or the PPARgamma-retinoid X receptor (PPARgamma-RXR) heterodimer is increased in combination with an increase in activity of at least one of PPARalpha and PPARdelta.
7. The method of claim 5 wherein said compound is administered a therapeutically effective amount sufficient to prophylactically prevent, slow, delay or treat at least one metabolic disorder or disease selected from the group consisting of insulin resistance, glucose intolerance, impaired glucose tolerance, impaired fasting serum glucose, impaired fasting blood glucose, hyperinsulinemia, pre-diabetes, type 1 diabetes, type 2 diabetes mellitus, insulin-resistant hypertension, the metabolic syndrome, the metabolic hypertensive syndrome, (metabolic) syndrome X, the dysmetabolic syndrome, obesity, visceral obesity, hypertriglyceridemia, elevated serum concentrations of free fatty acids, elevated serum concentrations of C-reactive protein, elevated serum concentrations of lipoprotein(a), elevated serum concentrations of homocysteine, elevated serum concentrations of small, dense low-density lipoprotein (LDL)-cholesterol, elevated serum concentrations of lipoprotein-associated phospholipase (A2), reduced serum concentrations of high density lipoprotein (HDL)-cholesterol, reduced serum concentrations of HDL(2b)-cholesterol, and reduced serum concentrations of adiponectin.
8. The method of claim 5 wherein the compound is of the general formula:



wherein R1 is an optionally substituted hydrocarbon residue which is optionally bonded through a hetero-atom; R2 is an optionally substituted 5-7 membered heterocyclic residue having, as a group capable of constituting the ring, a carbonyl group, a thiocarbonyl group, an optionally oxidized sulfur atom or a group convertible into them; X is a direct bond or a spacer having an atomic length of two or less between the ring Y and the ring W; W and Y are independently an optionally substituted aromatic-hydrocarbon residue

optionally containing a hetero-atom or an optionally substituted heterocyclic residue; n is an integer of 1 or 2; a and b forming the heterocyclic residue are independently one or two optionally substituted carbon or hetero atoms; c is an optionally substituted carbon or hetero atom; and, in the group of the formula:



substituents on adjacent two atoms forming the ring are optionally bonded to each other to form a 5-6 membered ring together with the two atoms forming the ring] or a pharmaceutically acceptable salt thereof, or a pharmaceutically acceptable solvate thereof.

9. The method of claim 8 wherein the compound is formulated for oral administration.
10. The method of claim 8 wherein the compound is formulated for topical administration.
11. The method of claim 9 wherein the compound is telmisartan, or an analog thereof.
12. The method of claim 11 wherein the total effective daily orally administered dose is selected from the range of about 20 mg to about 1000 mg.
13. The method of claim 9 wherein the compound is irbesartan, or an analog thereof.
14. The method of claim 13 wherein the total effective daily orally administered dose is selected from the range of about 20 mg to about 1000 mg.
15. The method of claim 1 wherein the mammal is a human child, adolescent or adult.
16. A method of screening compounds for treating or prophylactically preventing an inflammatory or metabolic disorder in a mammal, the method comprising selecting a compound that is able to:
 - (a) at least partially activate peroxisome proliferator activated receptors (PPARs); and
 - (b) at least partially inhibit, antagonize or block an activity of angiotensin II type 1 receptors.

17. The method of claim 16 further comprising selecting a compound that does not cause, promote, or aggravate at least one of fluid retention, peripheral edema, pulmonary edema, and congestive heart failure in the mammal.